Telephone numbers for the Editorial Office are:
(501) 296-1140
(501) 296-1141
(501) 296-1143
The FAX number is (501) 686-8124.
E-mail correspondence may be sent to: stnewell@medlan.uams.edu
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Errata

1. Schmidt S, Schöne N, Ritz E, and the Diabetic Nephropathy Study Group: Association of ACE gene polymorphism and diabetic nephropathy? *Kidney Int* 47:1176–1181.

In the article cited above, appearing in the April issue of the journal, Table 3B contains an error in the column Type I Diabetes With Nephropathy. The number of patients with ID should read 25, not 15. All calculations have been performed with the correct figure. The Editor and Publisher regret the error.

2. Martin P and Friedman LS: Chronic viral hepatitis and the management of chronic renal failure. *Kidney Int* 47:1231–1241.

In the article cited above, appearing in the May issue of the journal, the word "vaccine" was mistakenly omitted from the final sentence of the paragraph under the heading *Role of antiviral therapy in recipients of renal transplants* on page 1237. This sentence should read as follows:

The apparent inability of the host to mount neutralizing antibodies suggests that development of an effective HCV **vaccine** will be difficult [118].

The Editor and Publisher regret the error.

3. Textor SC, Burnett JC Jr, Romero JC, Canzanello VJ, Taler SJ, Wiesner R, Porayko M, Krom R, Gores G, and Hay E: Urinary endothelin and renal vasoconstriction with cyclosporine or FK506 after liver transplantation. *Kidney Int* 47:1426–1433.

In the article cited above, appearing in the May issue of the journal, a mechanical error caused part of page 1431 to be misprinted. The first few words in the first full paragraph of the righthand column on that page were deleted. The text of the paragraph is reproduced in full below.

Most importantly, our results establish that renal endothelin and prostacyclin excretion are disturbed after liver transplantation without direct manipulation of the kidney. These changes developed within a month after transplant and persisted indefinitely. Unlike studies in experimental animals, several human studies indicate that prostacyclin is suppressed during CsA administration [4]. We previously demonstrated this effect in the first several weeks after transplant during which time renal vasoconstriction and loss of GFR develops, despite rising arterial pressures and normalization of previously highly active renin-angiotensin system [11]. FK506 produces a similar hormonal and renal profile early after transplantation [3]. Results from the present study indicate that urinary endothelin increased simultaneously with the suppression of prostacyclin. Whether local release of ET caused the observed changes in prostacyclin, or *vice versa,* cannot be established with the present data. Nonetheless, these combined effects subject the renal vasculature to multiple hormonal disturbances favoring vasoconstriction. The presence of multiple simultaneous disturbances in the renal vasculature may explain the limited efficacy of maneuvers to enhance or block a single system, such as the administration of prostaglandin analogs (misoprostol [31]) or fish oil to shift the eicosanoid balance from thromboxane production [32].

4. Kaysen GA, Rathore V, Shearer GC, and Depner TA: Mechanisms of hypoalbuminemia in hemodialysis patients. *Kidney Int* 48:510–516.

In the article cited above, appearing in the August issue of the journal, the authors reported an error in Table 1, p. 513, left column. In this table, under the heading "Acute phase reactants," the units for α_2-macroglobulin and α_1-acid glycoprotein should be in milligrams per milliliter (mg/ml) rather than in milligrams per deciliter (mg/dl) with regard to the concentration of α_2-macroglobulin. These corrections do not change the interpretation of the data; however, they do correct an error in the report of concentration of these two proteins.